TABLE VI

No. 192-960. Age, 33 years

Date	Time in Coma	Acetone	Urine Sugar %	Blood Sugar	Insulin Units
July, 1923					
18	6 hrs.	2+	2.5	400	80
19		1+	2.5	400	80
20		0	2.0	500	140
23					
30		0	1.0	333	50
August 6		0	0	182	10
23		0	0	118	0

Discharged August 23, 1923. *No insulin.* Diet P. 50, C. 70, F. 205. This is the only patient in the series in whom insulin was discontinued.

TABLE VII

No. 201-942. Age, 22 years

Date	Acetone	Urine Sugar %	Blood CO_2 Vol. %	Blood Sugar	Insulin Units
January, 1924					
11	3+	3.3	10	400	150
12	3+	0	29.6	400	85
14		0.95	39.5	307	60
15		0.8	40.4	285	30
16		1.3	30.9	250	30
17		0.6	42.4	285	30
18		0.5	40.4	285	30
19		0.6	40.4	285	36
21		0.4	48.1	285	36
22		0.2	50.0	222	36
23	1+	0.4	50.0	222	45
24	0	0.08	50.0	200	45
28	0	0.09	—	181	36
Feb. 1	0	0.08	—	181	30

Discharged February 2, 1923. This patient has been under observation in the diabetic clinic during the past three years. He remains sugar free with a blood sugar of 250 mg. He receives 92 units of insulin daily. Any attempt to lower the blood sugar by increasing the insulin produces severe reactions.

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Large scale production, far-flung advertising, and the widespread distribution of goods and ideas have drained something of the color and flavor from American life. Men no longer react individually to conditions as they did in the isolation of the farm or the mine but respond to them under full cognizance of what their fellows are believing and feeling. They think alike, act alike, desire alike. It is a truism that deviation from the usual is nowhere more frowned down upon than in democratic America. Conformity is the price of respectability, and eccentricity is the deadly sin. As nowhere else in the world fashions and fads tyrannize in America.—*Saturday Review of Literature.*

Under the new Medical Practice Act of New York, podiatrists are forbidden to call themselves doctors, even though they follow this designation with the qualifying term of their craft. It is also prohibited to any but licensed physicians to use the expressions "foot specialist," "surgeon," "pedic surgeon," "orthopedic surgeon," or "orthopedic specialist."

PERFORATED ULCERS OF THE DUODENUM

TREATMENT BY HORSLEY OR MAYO PYLOROPLASTY

By EDMUND BUTLER* AND EVERETT CARLSEN

Simple closure is the rational procedure in perforations of acute duodenal ulcers. Such ulcers have existed only a very short time, and the patient often does not give a history of distress previous to perforation. Possibly about 20 per cent of perforated duodenal ulcers come under this classification.

Obstruction seldom follows closure of a perforated duodenal ulcer, therefore it is unnecessary to do a gastroenterostomy because of the fear of this complication.

The surgeon that occasionally encounters a perforated ulcer should be satisfied to do a simple closure.

Perforation of a chronic duodenal ulcer, if seen early, should be treated the same as perforating ulcers. If the induration is great and the patient's condition good a gastroenterostomy is advisable. If there is slight induration and the gastric wall in the region of the pylorus and the antrum is not too much indurated or thickened, then a pyloroplasty is indicated.

DISCUSSION *by R. W. Wilcox, Long Beach; Clinton D. Collins, Fresno; John Homer Woolsey, San Francisco.*

PATIENTS suffering from perforated duodenal ulcers present themselves for treatment in three stages. First the stage of contamination, secondly the stage of peritoneal reaction, and thirdly the stage of progressive peritonitis. During the stage of contamination continuous severe pain in the epigastrium and a board-like rigidity make a diagnosis of perforated ulcer most likely. During the stage of reaction the pain becomes less severe and the rigidity, although present, is less marked, the patient believes himself to be improving, thus the correct diagnosis may not be suspected and the decision to wait a few hours is frequently made. During the stage of progressive peritonitis it is difficult without a most carefully taken history to make a correct diagnosis. The diagnoses of cholecystitis, pancreatitis, diverticulitis, appendicitis, pyelitis and, in the female, pelvic inflammation must be ruled out.

The operative procedures depend on the stage in which the patient first comes for treatment. In the stage of contamination our procedure should be the same as though we were dealing with a perforating ulcer. The presence of a perforation within the first three or four hours should not deter the surgeon from doing a pyloroplasty or a gastroenterostomy. In the stage of reaction the operative procedure must be limited and simple closures and possibly pyloroplasty may be considered, most assuredly not a gastroenterostomy. During the stage of progressive peritonitis simple closure of the perforation is all that reasonably should be done, with pelvic drainage added in the latter stages. Drainage is not used in the stage of contamination or reaction, or in the early stages of peritonitis. Flushing of the peritoneal space should not be attempted.

The advice of some of our leading surgeons is confusing to one not sufficiently acquainted with this type of pathology to have developed considerable individual judgment. Guthrie summarized answers to questionnaires on the subject of perforation of duo-

* **Edmund Butler** (Medico-Dental Building, 490 Post Street, San Francisco). **M. D.** Cooper Medical College, 1911. **Practice** limited to Surgery. **Hospital connections:** San Francisco Emergency, Stanford, and St. Mary's and St. Joseph's hospitals. **Appointments:** Assistant Clinical Professor of Surgery, Stanford Medical School, and Child Surgeon, Emergency Hospital.

denal ulcers of 150 surgeons. Gastroenterostomy was done routinely by twenty-two, by sixty-four never, and in sixty-two a shifting percentage depending upon the condition of the patient, size and induration of the ulcer and the degree of stenosis. Deaver performs gastroenterostomy in every case. Richter is of the opinion that the perforation if properly closed would cause obstruction and therefore require gastroenterostomy. Gibson reports results not so good after gastroenterostomy as without it, and actual obstruction rarely occurs. Pool holds that routine gastroenterostomy is not advocated because with the average surgeon it increases the immediate risk. The mortality rate is little affected by gastroenterostomy, according to Hunt. K. Patterson Brown believes that gastroenterostomy gives a larger percentage of cures than does simple closure. Mills of England advocates routine gastroenterostomy. Wilensky is of the opinion that the most rational procedure in the presence of an acute perforation is to simply close the perforation. Stillman, my former chief, advocates simple closure and later a secondary gastroenterostomy may be necessary in a very low percentage of cases. Weeks, former chief of the San Francisco Hospital Service, is an advocate of simple closure, except when the ulcer is very near the pylorus and the closure is likely to produce obstruction, then a gastroenterostomy should be done.

Since December, 1919, sixty-eight patients suffering from perforated gastric or perforated duodenal ulcers have been operated upon in the Emergency Hospital Service. Of this series twenty-one have been simply closed. In thirty-seven gastroenterostomy was performed. In a recent series of ten perforated duodenal ulcers, Horsley-Mayo pyloroplasties have been performed. In the series of gastroenterostomies three patients were lost. In the series of simple closures seven patients were lost. Most of the patients that were lost following simple closure came in late and progressive peritonitis was well advanced.

At present we believe that pyloroplasty has a limited application, but we are not thoroughly convinced that the after results are any better following pyloroplasty than those that follow simple closure. In a subsequent paper we hope to be able to give data that will be more conclusive. Two patients of this series of pyloroplasties died, one had a sickle cell anemia, which has been previously reported by Gordon Hein of the San Francisco Hospital; the other developed a bronchial pneumonia the fourth day and died on the seventh day. No evidences of complications from the perforated ulcer were manifested.

The technique of the pyloroplasty is precisely that described by Horsley and by Mayo. Remember the structures are indurated and the procedure is more difficult, as the sutures will cut if too much tension is applied. The stomach must be pulled or shoved over to the duodenum as the temporary inflammatory induration fixes the duodenum. Incision must not extend the full two inches on the gastric side, as the opening of three or three and one-half inches in length would be most difficult to close transversely in the presence of the induration.

DISCUSSION

R. W. WILCOX, M. D. (114 East Seventh Street, Long Beach, California)—The most impressive factor of the treatise by Doctor Butler and Doctor Carlsen is the fact that perforations of the duodenum cannot be considered as a single entity with a routine operative repair for all cases. The individual, the age, the physical condition, the chronicity of the ulcer, and the time elapsing between the perforation and the time of repair, are factors that markedly alter the pathological picture.

The pathology found at operation determines the procedure that should be followed so as to give the patient first and above all else the best possible chance for recovery, and, second, the best possible functional result. The functional result may not be satisfactory; however, it is possible to correct this by further surgical procedure providing the patient is alive. The surgical judgment governed by the desire of the surgeon for the recovery of the patient, based on thorough pathological knowledge, will determine in each case the surgical procedure.

Artistic operations well performed are of no value if the patient does not recover.

Butler and Carlsen's classification of the stages of peritoneal involvement as a determining factor in the extent of surgical procedure is very good and gives the patient a maximum chance for recovery. The classification makes it axiomatic that the extent of surgical procedure decreases as the peritoneal invasion increases.

Simple closure of a perforation in a certain number of cases would give a lower mortality rate than a series of cases in equal number where more extensive operations were performed.

Secondary operations in a small percentage of cases following simple closure would be necessary, resulting, I believe, in a more satisfactory functional result than the result following immediate pyloroplasty or gastroenterostomy where the extent of pathology markedly handicaps the surgeon.

I have very much enjoyed the privilege of commenting on this excellent paper.

CLINTON D. COLLINS, M. D. (Mattei Building, Fresno, California)—The subject of treatment of perforation of gastric and duodenal ulcers has been well covered in this paper. The authors have given us a good working classification.

Most surgeons will agree, I believe, that in those patients who have reached the stage of progressive peritonitis, no more than a simple closure of the ulcer with or without drainage should be done. Even in the advanced cases, however, it may be necessary to excise the base of the ulcer before a satisfactory closure can be made. This is particularly true of indurated ulcers of the stomach.

In the earlier stages there is ground for difference of opinion. A factor which will often influence the extent of the operation and one which the authors have not emphasized is that of shock, which usually follows perforation of the stomach or bowel.

We often see patients in the earlier stages following perforation who are profoundly shocked. Unnecessary surgery should not be attempted under such circumstances. In these cases we are not dealing with ulcers of the stomach or duodenum per se, but with complications arising as the result of the perforation of the ulcer. Our efforts under these conditions should be directed, not so much toward curing the ulcer, but to carrying our patients safely through the immediate emergency. Experience has taught us that this end can be best accomplished by simple closure of the perforation, leaving more drastic curative measures to some future and more suitable time.

JOHN HOMER WOOLSEY, M. D. (490 Post Street, San Francisco)—The three stages of perforating duodenal and gastric ulcers are well chosen and defined. Agreement is also unanimously given to the authors' general outlined plan of treatment for each stage with the exception of the use of a pyloroplasty.

The advisability of a pyloroplasty, which obviously will be performed only in the first stage or period of contamination, is contrary to the experience at the University of California clinic. The first qualification for a

pyloroplasty is a freely movable, or, secondly, a mobilized duodenum. This prerequisite is as a rule not present in a chronic perforating duodenal ulcer, rarely present in an acute perforating duodenal ulcer, and a pyloroplasty never would be employed for a perforating gastric ulcer of the lesser curvature of the stomach. Mobilization by a lateral linear incision of the posterior peritoneum would be unwise in the presence of possible infection. Pyloroplasties performed where the duodenum is not freely mobile or where, as Butler and Carlsen say, "the stomach must be pulled or shoved over to the duodenum" do not give a good functional result and the majority have to come to further surgery.

It is recommended therefore that in the stage of contamination, simple closure of the perforating duodenal and gastric (pyloric antral) ulcer, with gastrojejunostomy only where obstruction is likely to ensue, and simple closure of the gastric ulcer of the lesser curvature with excision at times if possible but always accompanied by gastrojejunostomy, be performed.

CONTROL OF URINARY HEMORRHAGE*

By PAUL A. FERRIER *

DISCUSSION *by James R. Dillon, San Francisco; Franklin Farman, Los Angeles.*

WHILE the control of urinary hemorrhage is often simple, there are instances which tax the resources of the most skillful surgeon. It is proposed to consider these resources.

Successful treatment of hemorrhage includes the removal of the underlying pathological conditions. To recall the etiology, I have tabulated from MacKenzie, Kretchmer and Chute 1679 cases of hematuria, with their causes, in order of frequency.

	Cases	Per Cent
CAUSES OF HEMATURIA	1679	
KIDNEY	712	42.4
Pyelitis or pyelonephritis	217	12.3
Tuberculosis	180	10.7
Stone	145	8.6
Nephritis	56	3.3
Malignancy	54	3.2
Hydronephrosis	20	1.1
Trauma	12	.71
Polycystic kidney	11	.66
Congenital anomaly	11	.66
Pyonephrosis	4	.23
Syphilis	2	.11
Infarct	2	.11
Movable kidney	1	.05
Echinococcus	1	.05
BLADDER	527	31.3
Tumor	358	21.3
Stone	72	4.2
Chronic cystitis (simple)	49	2.8
Tuberculosis	14	.73
Hunner ulcer	9	.53
Diverticulum	8	.47
Trauma	7	.41
Polypi	3	.17
Cord bladder	2	.11
Fistula	2	.11
Cystitis cystica	1	.05
Angioma	1	.05
Ruptured artery	1	.05
PROSTATE	225	13.4
Hypertrophy	135	7.7
Cancer	61	3.6
Inflammation	13	.77
Tuberculosis	10	.58
Stone	4	.23
Foreign body	2	.11
URETER	151	8.8
Stone	142	8.4
Stricture	6	.35
Atony	2	.11
Cancer	1	.05
URETHRA	64	3.8
Urethritis	26	1.5
Trauma	13	.77
Stricture	12	.71
Caruncle	10	.58
Prolapse	2	.11
Polypi	1	.05

This list while not complete is representative. Essential renal hematuria of varied etiology is not tabulated, but is an important problem because of its differential diagnosis by exclusion, and its demand for control of bleeding.

In every hematuria diagnosis comes first. The initial bleeding is never fatal. Every day patients lost because the easy-going physician tides over a hemorrhage from tumor or tuberculosis while the opportunity for cure slips. Caspar states that in only three of 142 cases of bladder tumor seen soon after the first bleeding was the tumor large. By all means, diagnosis comes first. But help is often needed preliminary to radical treatment, and it is not always possible to remove the pathological lesions. Therefore, palliative hemostasis is important.

General as well as local causes must be considered. Clotting power may be at fault. While the mechanism of clotting is not fully understood, it is recognized that the clot is built upon fibrin, which exists in the blood as fibrinogen. Fibrinogen is coagulated by a protein substance thrombin, capable of being isolated and combining with 215 times its weight of fibrinogen. Thrombin exists in the blood as prothrombin, which is activated by tissue and blood cell juices called thrombokinase in the presence of a sufficient concentration of a soluble salt of calcium. The blood is kept from clotting in the veins by an antagonist to the prothrombin.

Four deficiencies may exist: that of calcium; of prothrombin; of platelets; of tissue juices, so called thrombokinase. The calcium clotting time may be tested and if calcium is lacking as, for example, in chronic jaundice, the administration of 10 cc. of 10 per cent calcium chloride intravenously on three successive days restores clotting. The common practice of giving calcium lactate by mouth is ineffective because of small absorption. In hemophilia prothrombin is lacking. In purpura platelets are low. These may only be supplied by blood or tissue juice from another person.

Clotting may be raised above normal by increasing the thrombokinase locally or generally. Locally by squeezing out the patient's own tissue juices. For example, Freyer advises massaging the prostatic capsule after enucleation; by heat; by transplanting muscle tissue, or by application of cephalin, or thromboplastin. For general action these may be injected subcutaneously. They have largely superseded horse serum on account of the danger of anaphylaxis and serum sickness.

Neuhoff and Hirshfeld found that intramuscular injection of sodium citrate greatly hastened clotting in normal or abnormal blood, except in platelet deficiency. The effect lasts two to three hours. Fifteen

* **Paul A. Ferrier** (Citizens' Savings Bank Building, Pasadena, California). **M. D.** University Pennsylvania, 1911. **Graduate study:** Mayo Clinic, 1915-18. **Practice** limited to Urology. **Hospital connections:** Urologist to Pasadena Hospital.